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18. (Currently amended) A method for inhibiting or reversing metastasis in a M+ class tumor, wherein said tumor is capable of existing in M+ and MO classes, comprising the step of contacting said with an effective amount and for an effective period of time with an inhibitor of the upregulation (overexpression) of a gene identified as being associated with said M+ class, said gene identification being made by a genetic method comprising the steps of:

A. Identifying by expression-profiling of tumor sample cohorts of said M+ and MO classes of said tumor, coupled with permutational statistical analysis, to generate a candidate gene list, those genes whose expression differ statistically between said classes of said tumor and that are upregulated in the M+ class and downregulated in the MO class;

B. producing a class-predictive algorithm based upon said predictive genes with a permutational *P* value of <0.05; and,

C. applying said algorithm to a candidate tumor to produce a Predictive Strength value that will assign the M+ or MO class to said tumor, wherein said algorithm comprises two primary equations:

$$(1)\ v_i = [\, x_i - (\mu_{Mo} + \mu_{M+}) / 2 \,]$$

wherein vi is the selective vote, xi is the expression level in the tumor sample, and μMO and μM+ are the metastatic classes of reference samples, and wherein said votes are summed in order to obtain total votes for the non-metastatic (V_{Mo}) and metastatic (V_{M+}) classes; and,

$$(2)\ \text{Prediction Strength} = [\, (V_{Mo} - V_{M+}) / (V_{Mo} + V_{M+}) \,]$$

wherein Prediction Strength values range between 0 and 1.

19.(Currently amended) The method according to claim 26, wherein said inhibitor is a neutralizing antibody directed against the protein encoded by said upregulated M+ gene.

20.(Currently amended) The method according to claim 26, wherein said inhibitor is a chemical inhibitor.

21. (Original) The method according to claim 20, wherein said inhibitor is directed against a member of the the metastatic overexpressed gene group consisting of the signal transduction inhibitor STI-571, the *RAS* inhibitor R115777, the MAP2K1/MAP2K2 protein kinase inhibitor U0126, the specific signal transduction inhibitor of PDGFRA STI-571, the phosphoinositide 3-kinase inhibitor wortannin, the *VEGF* inhibitor NM3, the *MAP* kinase inhibitor CC1-779, and the glutathione S-

transferase inhibitor TLK 886 .

22. (Original) The method according to claim 21, wherein said inhibitor is the *RAS* inhibitor R115777.

23. (Original) The method according to claim 21, wherein said inhibitor is SCH88336.

24. (Original) The method according to claim 21, wherein said inhibitor is U0126.

25. (Original) The method according to claim 21, wherein said inhibitor is STI-571.

26 (New) The method of claim 18, wherein said upregulated tumor gene is the gene for PDGFRA or a gene downstream from said PDGFRA gene.

27 (New) The method of claim 26, wherein said downstream gene is selected from the group consisting of RAS, MAP2K1/MAP2K2, phosphoinositide-3-kinase, VEGF, MAP kinase, and glutathione-S-transferase.